Small Bowel Rupture from Abdominal Blows

HARRY PERELMAN, *M.D.*, Los Angeles

• **Rupture of the small bowel with nonpenetrating abdominal trauma occurs in one-third to one-fourth of all abdominal injuries. Perforation of the bowel can result from injuries of crushing, bursting or tearing type. The symptoms are those associated with peritoneal irritation. Repeated observation and examination is necessary for diagnosis. Operative repair is mandatory. Two cases, one in a child, are reported.**

RUPTURE of almost every organ within the abdominal cavity has occurred without external signs of injury. Injuries to the kidneys, spleen, liver and pancreas are the most common, these being solid, fixed abdominal viscera. From one-quarter to one-third of all abdominal injuries involve the small bowel alone.[2,8,11] Cottrell[1] noted that the intestine was injured in 10 per cent of 800 cases he reviewed. Trauma is produced by blows, falls, blasts, car collisions and similar accidents. Automobile collision is by far the most common cause of nonpenetrative abdominal trauma, impact with the steering wheel (which is also an etiological factor in liver trauma)[9] being noted frequently. The fragility of the small bowel and the fact that it can be ruptured by trauma that does not injure the skin have been recognized since the time of Aristotle. It was a common cause of death in the Middle Ages, often from kicks of horses or blows.[7]

Small bowel rupture occurs most commonly where the bowel is either firmly fixed to the posterior abdominal wall or has a short mesentery—these sites being the duodenum, the jejunum near and immediately distal to the ligament of Treitz, and the ileum just proximal to the ileocecal junction. The side most often involved is the antemesenteric border. Multiple lacerations of the small bowel occur in 10 per cent of cases.[1,3,5,12]

MECHANISM OF THE RUPTURE

Geohegan[3] expressed belief that the majority of ruptures of the small bowel from nonpenetrating trauma are bursting injuries caused when a sudden force is applied to the abdominal wall and the increase in the intra-abdominal and intraperitoneal pressure is transmitted to the hollow viscera. He postulated that the length and tortuosity of the intestinal tube is such that the sudden increase in pressure in the mid portion may not be dissipated by release through its open ends and the pressure, reaching a maximum at a point near a turn or kink which provides a trap for the fluid and gas, causes rupture there. Poer and Woliver[10] said that most frequently such perforations result from a crushing injury in which the external force compresses the bowel against the spine or pelvic bones, but that sometimes tearing injuries may result from a violent force being applied at a tangent to the body.

From the Department of Surgery, Culver City Hospital, Culver City.
Submitted August 31, 1960.
This study was aided by a grant from the Isaac Brotman Foundation of the Culver City Hospital.

SYMPTOMS

Perforation of a hollow viscus must be suspected when there is persistent abdominal pain, spasm and tenderness, either with or without nausea or vomiting, after severe trauma to the abdomen. Signs of peritoneal irritation, with local or general abdominal tenderness and rigidity, are present. The higher the perforation, the more likely that chemical irritation is the cause of peritonitis; the lower the perforation, the more likely that peritonitis is bacterial.[6] Peristalsis is either absent or greatly diminished. Pain is severe, continuous and generalized. Pain referred to the shoulder or neck is indicative of diaphragmatic irritation due to either free air or escaped fluids. Pain referred to the rectum and perineum with associated tenesmus is frequently a sign of high rectal or sigmoid injury. Pain may be referred to the right testis as a result of irritation of the genitofemoral nerve by spillage from retroperitoneal rupture of the duodenum. A very significant character of peritoneal pain due to spilling of either hollow visceral contents or blood is the increase that occurs when the patient moves. Hemorrhage is not an outstanding feature.

Complete rupture may be single or multiple and may be associated with mesenteric contusions, tears or detachments; in such instances there will also be severe hemorrhage. Localized signs are commonly absent for three to twelve hours.

The pulse rate increases as soiling of the peritoneum progresses. It is also increased in hemorrhage and shock and is therefore significant of serious intra-abdominal injury.

Except in infants, gas is not normally roentgenographically visualized in the small intestine. Therefore, in perforations of the healthy small intestine, evidence of free intraperitoneal gas is the exception rather than the rule; seldom is there evidence of intraperitoneal air in cases of jejunal and ileal perforation. An exception is retroperitoneal duodenal rupture, in which retroperitoneal air may be seen to outline the psoas muscle shadow. However, free gas may be demonstrated in approximately 80 per cent of patients with perforation of the stomach or the first portion of the duodenum, and in almost all of those with perforation of the colon.[4]

The presence of free blood in the stomach indicates upper abdominal injury. If fresh or darkened blood is seen on rectal examination, intestinal injury is likely.

The leukocyte count becomes rapidly elevated in most instances of intraperitoneal hemorrhage and also in cases of chemical peritonitis from upper gastrointestinal spillage. The erythrocyte count is not dependable as an index of loss of blood. Hemoglobin and hematocrit determinations, as single determinations, are misleading.

All patients with a history of abdominal trauma should be closely observed up to 48 hours, with repeated examinations of the abdomen, pulse, blood pressure, respiration and hemoglobin and leukocyte count. There may be a high coincidence of other severe injuries such as multiple fractures and craniocerebral trauma, with the patient perhaps comatose, or complications such as alcoholic intoxication. Such complications greatly increase the difficulty of evaluating an abdominal lesion requiring immediate operation.

TREATMENT

Surgical treatment is mandatory as soon as the diagnosis is made. The mortality rate increases with lengthening of time between rupture of the bowel and operation. The rate for jejunal perforations is twice that for ileal perforations.

Intestinal decompression and administration of antibiotics should be carried out, together with indicated blood and fluid replacement in preparation of the patient for operation. Spinal anesthesia was the most successful anesthesia in a series reported by Geohegan.[3] At operation, thorough exploration of the abdomen must be carried out even after one perforation is located, for often there are multiple perforations and other intra-abdominal injuries. The perforation in the bowel can be treated by either simple closure or by resection, depending on the extent of the involvement, with anastomosis if necessary. All contaminating material should be washed from the abdomen.

REPORTS OF CASES

CASE 1. A 5-year-old boy ran into a parked automobile while running down a hill. This was at about 3:00 p.m. He went home complaining of severe pain in the abdomen. He vomited blood and soon afterward was taken to the office of a physician. The abdomen was rigid but not board-like. There was tenderness across the upper portion. Resonance to percussion over the liver was noted. The patient was admitted to the hospital at 5:30 p.m.

X-ray films of the abdomen showed free gas beneath the domes of the diaphragm. Leukocytes numbered 17,000 per cu. mm.—64 per cent segmented forms, 18 per cent stabs and 18 per cent lymphocytes. The hemoglobin content was 14.4 gms. per 100 cc. Erythrocytes numbered 4.53 million per cu. mm. The body temperature on admission was 98.8° F. and the pulse rate was 100 per minute.

At laparotomy a perforation 1.5 cm. in diameter was found in the jejunum 20 to 25 cm. distal to the ligament of Treitz. The ruptured area was punched out and rounded and allowed free exit of the intestinal contents into the peritoneal cavity. There was a large amount of plastic exudate over the surface of all loops of small bowel. The perforation was repaired, the surrounding area was washed with copious amounts of saline solution and the abdomen was closed. The body temperature was elevated for about a week after operation, then subsided. The patient did well thereafter.

CASE 2. A 49-year-old man was struck across the lower abdomen by a piece of 2 x 4 wood that was flipped from the rapidly whirling blade of a power saw. The time of the accident was about 9 a.m. The patient continued to work that morning but went home in the afternoon. There he vomited one to two quarts of black liquid. He had severe aching across the lower abdomen and great aggravation of abdominal pain when he moved. Soon after examination in the office he was admitted to the hospital, at 7:42 p.m. The pulse rate was 120 per minute, and the blood pressure 140/80 mm. of mercury. Board-like rigidity and pronounced tenderness of the entire abdomen were noted. There was decreased dullness to percussion over the liver, bilateral shifting flank dullness and questionable evidence of fluid wave. On auscultation bowel sounds were absent.

X-ray films of the abdomen showed free gas under the domes of the diaphragm.

Leukocytes numbered 11,900 per cu. mm.—80 per cent segmented forms, 15 per cent stabs and 5 per cent lymphocytes. The hemoglobin content was 16.7 gm. per 100 cc. Erythrocytes numbered 5.4 million per cu. mm. The rectal temperature was 100.2° F. Results of urinalysis were within normal limits except for the presence of acetone.

A Miller-Abbott tube was inserted and the patient was taken to surgery. At laparotomy there was a plastic exudate over all loops of the small intestine and a large collection of brownish-green fluid in the pelvis. A perforation 0.5 cm. in diameter with ragged edges was found in the antemesenteric border of the ileum, 25 cm. proximal to the ileocecal valve. The entire small bowel was examined from the ligament of Treitz to the ileocecal area and no other perforations or evidence of trauma were found. The perforation was closed and incidental appendectomy was done. After the abdominal cavity was irrigated with copious amounts of saline solution, the abdomen was closed except for Penrose drains that were brought out through a stab wound. The patient was discharged from the hospital nine days after operation and he was well thereafter.

6010 Wilshire Boulevard, Los Angeles 36.

REFERENCES

1. Cottrell, J. C.: Nonperforative trauma to abdomen, Arch. Surg., 68:241-251, Feb. 1954.

2. Estes, W. L. Jr., Bowman, T. L., and Meilicke, F.: Nonpenetrating abdominal trauma with special reference to lesions of the duodenum and pancreas, Am. J. Surg., 83: 434-452, March 1952.

3. Geohegan, T., Gordon, E. J., and Broch, B. E.: Small intestinal rupture from nonpenetrating abdominal trauma, J. Michigan M. Soc., 54:1223-5, Oct. 1955.

4. Jacobson, G., and Carter, R. A.: Small intestinal rupture due to nonpenetrating abdominal injury; a roentgenologic study, Am. J. Roentgenol., 66:52-64, July 1951.

5. Kirtland, H. B., Jr.: Rupture of the gastrointestinal tract; dealing with lesions caused by nonpenetrating trauma, Calif. Med., 79:434-6, Dec. 1953.

6. Kulowski, J.: Crash Injuries, Charles C. Thomas, Springfield, Ill., 1960.

7. Newing, R.: Rupture of small bowel in association with procidentia, M. J. Australia, 42:701-2, Nov. 26, 1955.

8. Patterson, R., and Bromberg, B.: Abdominal injuries with special reference to errors in early handling, Am. J. Surg., 83:427-433, March 1952.

9. Perelman, H., and Sherwood, H. R.: Injury to the liver in automobile accidents, J.A.M.A., 173:1019-20, July 2, 1960.

10. Poer, D. H., and Woliver, E.: Intestinal and mesenteric injury due to nonpenetrating abdominal trauma, J.A.M.A., 118:11-15, Jan. 3, 1942.

11. Pontius, G. V., Kilbourne, B., and Paul, E.: Nonpenetrating abdominal trauma, Arch. Surg., 72:800-811, May, 1956.

12. Williams, E. R.: Abdominal trauma; rupture of the jejunum and hemopneumothorax following nonpenetrating trauma, J. Kansas M. Assn., 58:446-7, June 1957.